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Life-threatening pericarditis and pleuritis caused by influenza B and successfully treated during COVID-19 pandemic

Zagrażające życiu zapalenie osierdzia i opłucnej spowodowane przez grypę B i skutecznie wyleczone podczas pandemii COVID-19

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Abstract

In 2020, World Health Organization declared severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection [coronavirus disease 2019 (COVID-19)] a pandemic. Nowadays, Influenza A (InfA) and COVID-19 seem to be a major threat. We report the rare case of life-threatening exudative pericarditis (without myocardial involvement) and pleuritis in an adult patient without inherited diseases, caused by Influenza B (not by COVID-19/InfA) and successfully treated (including: oseltamivir, pericardiocentesis 700 mL, pleurocentesis 1100 mL) in the COVID-19 pandemic. Although Influenza B is a rare cause of pericarditis, we should consider it as a possible reason of a potentially lethal disease.

Key words: influenza B, pericarditis, pleuritis, COVID-19 pandemic, SARS-CoV-2

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Introduction

Influenza causes 300,000 deaths per year in the world [1]. Although the respiratory system is commonly affected, cardiac involvement also occurs (direct virus impact on heart or preexisting cardiovascular disease exacerbation) [2]. In 2020, World Health Organization (WHO) declared SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) infection (COVID-19, coronavirus disease 2019) a pandemic. Influenza A (InfA) and COVID-19 seem to be a major threat. Influenza B (InfB) is considered less severe than InfA, typically causing mild upper respiratory symptoms [3].

Authors report the rare case of life-threatening pericarditis (without myocardial involvement) and pleuritis, caused by InfB and successfully treated during a COVID-19 pandemic.

Case report

A 74-year-old patient with I/II/III degree atrioventricular block, after pacemaker implantation (19.02.2020) and right ventricular (RV) electrode replacement (dysfunction, 21.02.2020), was admitted to hospital (19.03.2020) because of confirmed (Doppler-ultrasound) left brachial, axillary vein thrombosis and fever (up to 38°C) for 7 days.

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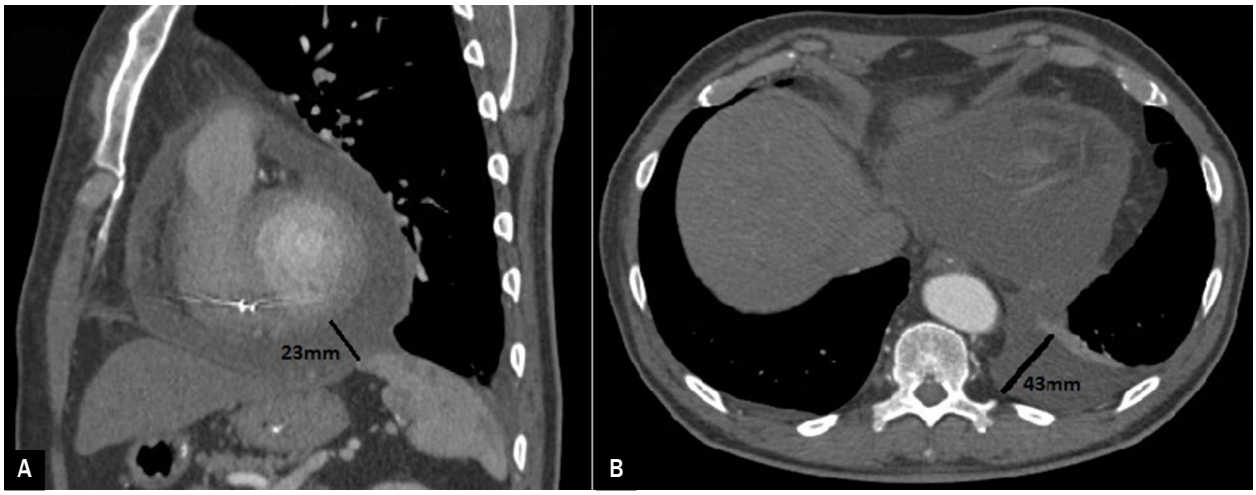

Figure 1A. Chest computed tomography (CT): fluid in pericardium 23 mm; **B.** Chest CT: fluid in left pleura 43 mm

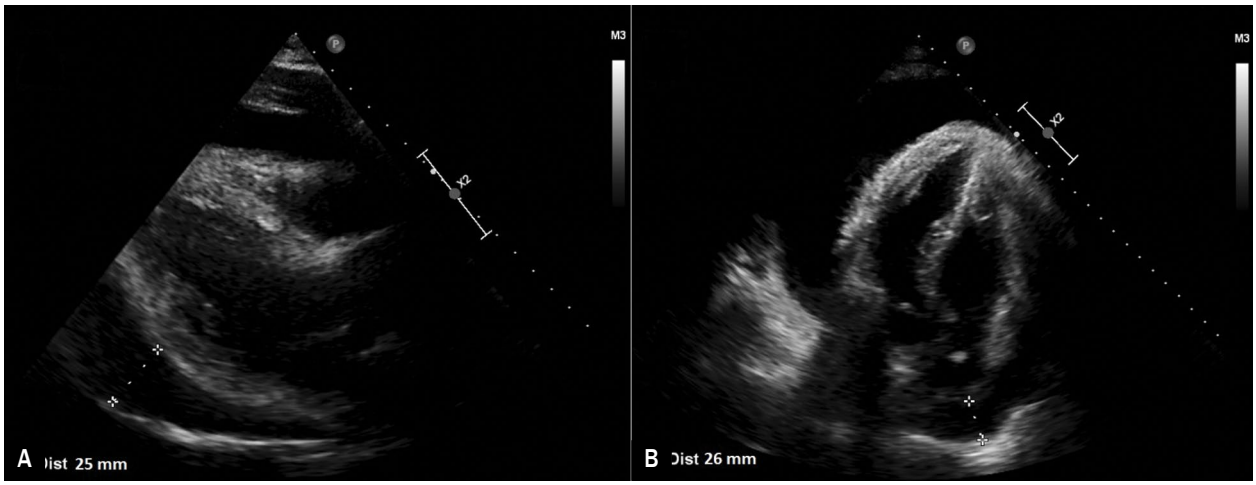

Figure 2. Transthoracic echocardiography prior to pericardiocentesis: pericardial effusion at right ventricle 15 mm, posterior wall of left ventricle (LV) 25 mm, lateral wall of LV 27 mm, right atrium 26 mm: **A.** Long axis view; **B.** Four chamber apical view

Initially, the patient's state was good, without dyspnea, stenocardia, cough; 36.8°C, the pacemaker pocket healed properly. Upper left extremity was swollen, warm.

COVID-19 was excluded. "Cassette" screening test and reverse transcriptase polymerase chain reaction (RT-PCR) (nasopharyngeal swab, days: 1, 14) were negative.

Methicillin resistant *Staphylococcus aureus* (MRSA), *Klebsiella pneumoniae* carbapenemase (KPC) (swabs), tuberculosis (QuantiFERON) were excluded. Inflammatory markers elevated: C-reactive protein (CRP) 205 mg/L, D-dimers 0.69 mg/L FEU, white blood cells WBC 12,000/μL, with negative procalcitonin.

Cardiac device-related infectious endocarditis (CDRIE) was suspected. Transthoracic echocardiography (TTE) revealed pericardial effusion at RV 5 mm, no systolic RV and left ventricular (LV) dysfunction. Transesophageal echocardiography (TOE) showed no vegetations. Pharmacotherapy was provided: ointment (heparin, phenyl butasone), rivaroxaban 2 × 15 mg, ketoprofen, and empirical treatment with vancomycin. Inflammatory markers decreased, but hectic fever (to 38.5°C) of unclear etiology persisted.

Urine cultures and tumor markers [CA125, CA19.9, carcinoembryonic antigen (CEA), CA15.3, prostatic specific antigen (PSA)] were negative. Abdominal computed tomography (CT) revealed fluid in pelvis 17 mm; chest CT fluid in pericardium 23 mm, in left pleura 43 mm (Figure 1). TTE showed fluid at RV 5 mm, LV posterior wall (LVPW) 11 mm, right atrium (RA) 11 mm, without wall perforation.

Proteinogram confirmed acute inflammation. Hypothyroidism was excluded.

Blood cultures were negative. TOE was repeated (days 1, 7): no vegetations, pericardial effusion 22 mm, without tamponade, 5 mm fibrin layer. There was no data for CDRIE. Initial diagnosis was reactive/inflammatory pericardial and pleural effusion. Colchicine, ibuprofen, levofloxacin were administered.

Control Doppler-ultrasound (after week) showed residual 1.5–2.0 mm thick wall thrombus in left axillary veins. Rivaroxaban dose was reduced (20 mg/day).

After 10 days of treatment: clinical worsening, still fever, increasing CRP (238 [mg/l]), WBC (14,500/μL), D-dimers (7.89 mg/l FEU)) were observed. Paroxysmal atrial fibrillation/atrial tachycardia appeared (after fluid evacuation: spontaneous sinus rhythm recurrence). TTE showed pericardial effusion at RV 15 mm, LVPW 25 mm, RA 26 mm (Figure 2).

Nasopharyngeal swab samples were negative for InfA, positive for InfB. Oseltamivir 2 × 75 mg and furosemide were provided (control swab after 5 days: negative).

Due to pericardial and pleural (82 mm) effusion, pericardiocentesis (700 mL, serosanguinous fluid) and pleurocentesis (1100 mL, serous fluid) were performed (02.04.2020), with clinical improvement. Pericardial fluid examination revealed exudate, negative cultures (aerobic, anaerobic, fungal, TBC). Pleural fluid analysis showed exudate, negative cultures (aerobic, anaerobic, TBC).

Anti-cardiolipin antibodies (ACA: IgM, IgG), anti-nuclear nRNP, anti-neutrophil (cANCA, pANCA), antinuclear and cytoplasmic ANA (HEP-2), *Legionella, Bartonella, Tularemia* antibodies were negative. *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* IgM antibodies were negative, IgG were positive (previous infections). Prednisone (30–100–0 mg) was provided.

Finally, clinical improvement, no fever recurrence and CRP reduction (15) were achieved. Control TTEs revealed stable pericardial effusion at LVPW 18 mm, RA 13 mm. The patient was discharged with diagnosis of exudative pericarditis and pleuritis in the course of InfB.

Follow-up: continuous decrease in pericardial effusion.

Discussion

Cardiac involvement is more common and better described with InfA than InfB [3].

Few cases of myopericarditis were described in the course of: COVID-19 (pericardiocentesis 540 mL) [4], and InfB (fatal tamponade, pericardiocentesis 240 mL) [3].

Only few cases of pericarditis (without myocardial involvement) were reported, but in "high risk" patients with co-morbidities, during: InfA (first case: hypertension, nicotinism; pericardiocentesis 250 mL, ibuprofen, colchicum, oseltamivir [2]; second case: alcohol, cocaine; pericardiocentesis, antibiotic, oseltamivir, nonsteroidal anti-inflammatory drugs (NSAIDs), colchicum [5]) and InfB (21 trisomy, ASD; ibuprofen, colchicum, oseltamivir, steroids, furosemide) [1].

We report a rare case of life-threatening exudative pericarditis (without myocardial involvement) and pleuritis in an adult patient without inherited diseases caused by InfB (not by COVID-19/InfA) and successfully treated in a COVID-19 pandemic. However, InfB is a rare cause of pericarditis we should consider it as a possible reason of a potentially lethal disease.

Funding

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Conflict of interest

The authors declare no conflict of interest.

Streszczenie

W 2020 roku Światowa Organizacja Zdrowia ogłosiła panedmię spowodowaną SARS-CoV-2 (*severe acute respiratory syndrome coronavirus 2*) (choroba koronawirusowa 2-19 [COVID-19]). Infekcje grypą A (InfA) i COVID-19 są obecnie traktowane jako większe zagrożenie niż infekcja grypą B. Zaprezentowano rzadki przypadek zagrażających życiu wysiękowego zapalenia osierdzia (bez zajęcia miokardium) i opłucnej u dorosłego pacjenta (bez chorób dziedzicznych), wywołanych przez grypę B (nie przez COVID-19/InfA) oraz skutecznie wyleczonych (w tym: oseltamiwir, perikardiocenteza 700 ml, pleurocenteza 1100 ml) w czasie pandemii COVID-19. Mimo że grypa B jest rzadką przyczyną zapalenia osierdzia, to należy ją traktować jako możliwą przyczynę potencjalnie śmiertelnej choroby.

Słowa kluczowe: grypa B, zapalenie osierdzia, zapalenie opłucnej, pandemia COVID-19, SARS-CoV-2

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